Pulmonary Collapse as a Complication of Bronchial Asthma in a Child

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ALTHOUGH MASSIVE pulmonary atelectasis following bronchial asthma is rare in childhood it occurs often enough to be kept in mind in order that preventive measures may be undertaken.

REPORT OF A CASE

A boy seven and a half years of age who had had asthma since age three was examined January 14 because of pain low in the left side of the chest and the development of scoliosis to the right.

The illness had begun January 9 with a cough and audible wheeze, for which his mother gave him several doses of Quadrinal,® a preparation containing ephedrine, phenobarbital, theophylline-calcium salicylate and potassium iodide, which had seemed to relieve his asthma in the past. Then, having run out of this drug, she gave him an antihistamine which had once been given to the patient for urticaria. She reasoned that, since she had been told his hives were on an "allergic basis" the antihistamine might also relieve the asthma. The patient became nauseated and the mother had difficulty in getting him to take liquids. It was on January 13 that right scoliosis and thoracic pain developed.

When examined on January 14, the patient had no pain in the chest and he did not look very ill. The color of the skin was good. The oral temperature was 98.8°F. and the pulse rate 140. Moderate dyspnea was noted. No audible wheeze was present. The patient did not cough during the examination. There was pronounced right scoliosis. Breath sounds were diminished over the lower left lobe and there was a shift of the mediastinum to the left. No wheezes or rales were heard on auscultation. The hemoglobin content was 15.3 gm. per 100 cc. of blood. Erythrocytes numbered 5 million per cu. mm. and leukocytes 10,400 per cu. mm.—59 per cent neutrophils (segmented 56 per cent and band forms 3 per cent), 25 per cent lymphocytes, 12 per cent monocytes, and 4 per cent eosinophils. Results of urinalysis were within normal limits.

X-ray films of the chest showed diffuse homogeneous pleural densities at the left bases. The left diaphragm was elevated, the mediastinal structures were retracted to the left and the costal interspaces narrowed. The findings were consistent with atelectasis of the left lower lobe and associated pleural reaction. Except for pleural densities, the left upper lung field was clear. The right lung showed only some increase in peribronchial markings at the right base.

A consultant who examined the patient concurred in the impression of left lower lobe atelectasis, most likely owing to plugging of the left main bronchus with inspissated mucus.

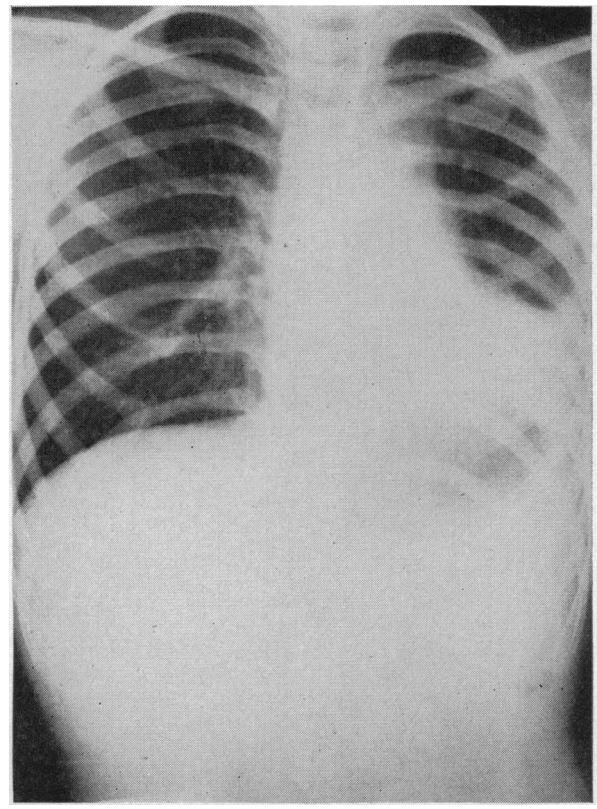

Figure 1.—Roentgenogram of chest on January 14, 1958, showing left lower lobe atelectasis, elevation of diaphragm and mediastinal shift.

Orthoxicol,® a preparation of dihydrocodeinone bitartrate, methoxyphenamine and sodium citrate, was given for expectorant and bronchial dilation effect, and daily inhalations of isoproterenol hydrochloride and oxygen were administered with an intermittent positive pressure breathing apparatus. V-Cillin K* was given, one 125 mg. tablet three times a day.

X-ray films on January 17 indicated persistence of the left lower lobe atelectasis but showed improved aeration and some regression of the pleural reaction.

Although the patient remained afebrile and quite comfortable, it was believed that bronchoscopic examination would have to be done unless there was pronounced improvement within 48 hours. By that time, however, stethoscopic examination indicated better aeration of the left lower lobe. On January 24 the patient was feeling better and was still afebrile. An x-ray film showed complete reexpansion of the left lower lobe with no evidence of atelectasis.

The patient was permitted full activity and returned to school January 27.

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*A penicillin preparation (Lilly) for oral administration.

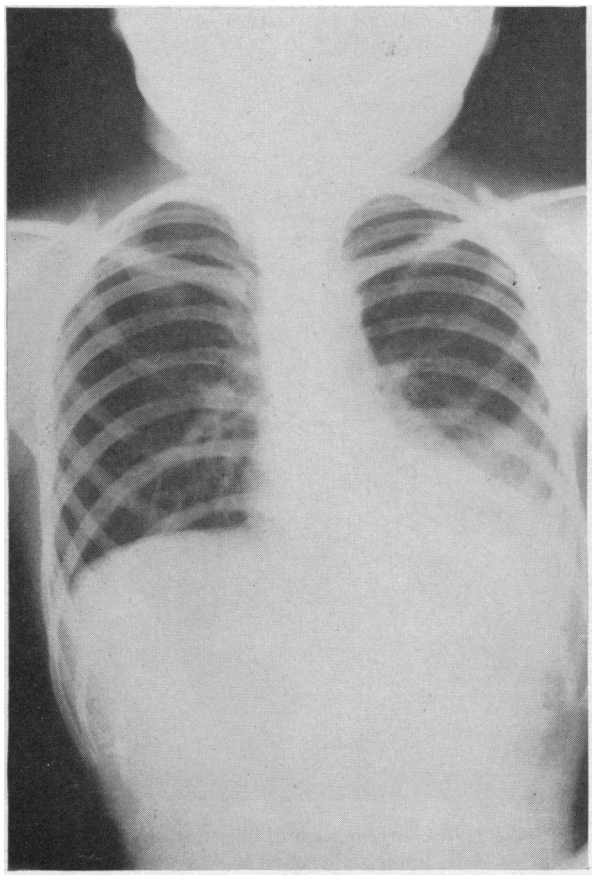

Figure 2.—Roentgenogram of chest on January 17, 1958, showing improved aeration in lower left lobe.

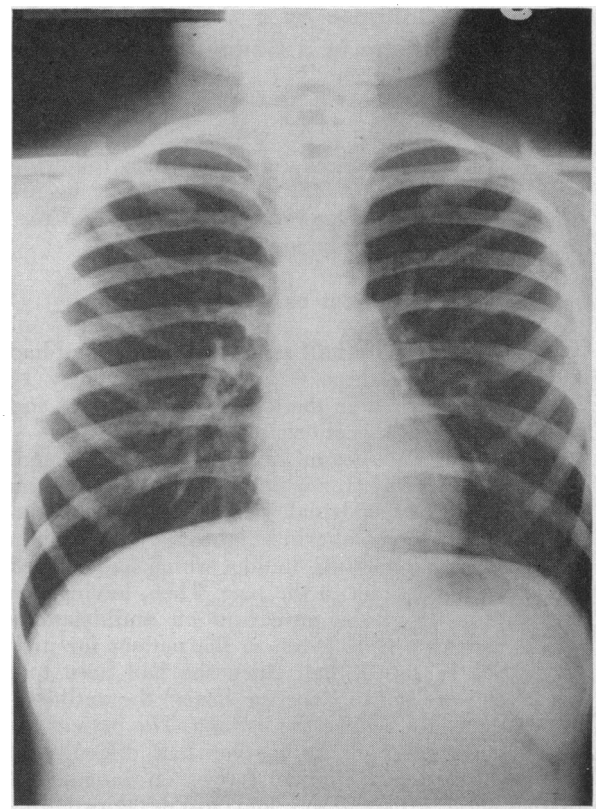

Figure 3.—Roentgenogram of chest on January 24, 1958, showing complete expansion of lower left lobe and normal position of diaphragm and mediastinum.

DISCUSSION

In a study of 854 children with pulmonary collapse James and co-workers[3] noted that 56 cases or 6.5 per cent were owing to bronchial asthma. Sixty per cent of the 56 patients were under five years of age. In 25 per cent of the cases the condition recurred.

According to Rakower and co-workers,[4] massive atelectasis is a rare complication of bronchial asthma and it occurs more frequently among children than adults because the smaller caliber of the bronchi in childhood predisposes to bronchial obstruction. These observers also noted that accompanying and following an attack of bronchial asthma there is edema of the bronchial mucosa with increase in bronchial mucus. At the same time there is a decrease in the ciliary action, thereby promoting stasis of secretions. Dehydration from loss of fluid during increased respiratory activity promotes drying of the mucus into inspissated plugs that obstruct the bronchi. Removal of these plugs is usually followed by prompt clinical improvement.

Glaser[1] said that the most common error in the treatment of status asthmaticus in childhood is failure to keep the patient well hydrated, which results in plugging of the smaller and sometimes larger divisions of the bronchi with mucus and may cause death from suffocation. He said that administration of adequate fluids, cold steam and expectorants and the avoidance of drying agents such as atropine and antihistamines are of paramount importance in the prevention of bronchial plugging.

Hinshaw and Garland[2] said that not only are antihistaminic drugs of very little value in the treatment of bronchial asthma but they have an atropine-like drying effect which, by making the mucus thicker and more tenacious, adds to respiratory difficulty. From the foregoing it can be summarized that treatment for the prevention of atelectasis due to mucus bronchial plugging in bronchial asthma should include adequate hydration, use of expectorants, bronchial dilation and caution in the use of antihistaminic drugs if they are used at all. In addition, the author also prescribes antibiotics, especially if fever is present.

In dealing with a sick child, it is usually difficult to maintain adequate fluid intake by mouth. An easy and effective substitute is retention enemas of one-half normal saline content, given in amounts of 120 to 240 cc. (in portions of 60 cc. every half hour) every 4 to 6 hours to make up the fluid deficit. This may obviate the need to put the patient in a

hospital. The parents are instructed to note the color of the urine. If it is pale straw or lighter, hydration is adequate.

SUMMARY

A case of bronchial asthma in a boy seven and a half years old, complicated by a massive pulmonary atelectasis, is presented. The immediate cause was thought to be plugging of a main bronchus with inspissated mucus, enhanced by dehydration and the injudicious use of an antihistaminic drug by the patient's mother.

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Spontaneous Pneumothorax Complicating Pulmonary Metastasis of Sarcoma

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SPONTANEOUS PNEUMOTHORAX is an unusual complication of pulmonary metastasis from sarcoma. Only 15 cases with this association have been reported previously. Sherman and Brant[6] reported six such cases and also a case of metastatic adenocarcinoma (primary site undetermined) in which spontaneous pneumothorax developed. In eight of the 16 cases there was metastasis to both lungs. Thirteen of the patients were male and three female. The age range was from 9 to 55 years, but only three patients were more than 23 years of age. The tumors from which pulmonary metastasis originated were as follows: Osteogenic sarcoma in five cases, Ewing's tumor in three, fibrosarcoma and leiomyosarcoma in two each, and adult Wilms' tumor, rhabdomyosarcoma, angiosarcoma and metastatic adenocarcinoma (primary undetermined) in one case each.[1,3,4,5,6,7]

Although Sherman and Brant[6] believed the case they observed of metastatic adenocarcinoma was the first to be reported in association with pneumothorax, Heimlich and Rubin[2] reported three cases in which spontaneous pneumothorax was a presenting feature of primary lung cancer. This association is relatively rare, however, and there seems to be a more particular tendency for sarcoma to produce pneumothorax.

In discussions of pathogenesis Thornton and Bigelow[7] and Lodmell and Capps[4] expressed belief that not all cases come about in the same way. In some cases pneumothorax results from necrosis of a subpleural nodule with formation of a bronchopleural fistula. In others it may arise from bronchiolar obstruction by tumor, which brings about alveolar ectasia and interstitial emphysema, which in turn (by the mechanism described by Macklin and Macklin) causes air to travel by way of perivascular connective tissue spaces toward the mediastinum or visceral pleura.

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REPORT OF A CASE

A 15-year-old white girl had a high thigh amputation in June of 1951 for osteogenic sarcoma of the distal femur. About six months later malaise and fatigue developed and then a hacking cough. After two weeks, severe cough and dyspnea required hospital admission on January 25, 1952. A film of the chest taken with a portable x-ray machine showed bilateral pneumothorax with collapse of about 40 per cent of the right lung and 25 per cent of the left lung. Metastatic lesions in both lungs ranged from 0.5 to 3 cm. in diameter, and there was left pleural effusion. The patient was treated with oxygen, codeine and antibiotics and was somewhat relieved. A film of the chest on February 13 showed a massive left pleural effusion, but pneumothorax was no longer present. The condition of the patient deteriorated rapidly and she died on March 22. Autopsy was not done.

SUMMARY

A case of spontaneous bilateral pneumothorax complicating osteosarcoma with pulmonary metastasis is reported and the literature reviewed.

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